Original Research

Glucomannan From Porang (Amorphophallus muelleri) Improves Short-Chain Fatty Acid in Wistar Rat with High-Fat and High-Fructose Diet

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Introduction: Short-chain fatty acids (SCFAs) improve lipid profile and prevent coronary artery disease. Searching for nutrition based on local foods that might raise the body's SCFA levels is imperative. Porang (Amorphophallus muelleri) is a plant with a high concentration of glucomannan that is investigated to have a healthy benefit. This study aimed to investigate the effect of glucomannan from Porang (A. muelleri) on SCFA in Wistar rats with a high-fat, high-fructose diet.

Methods: This was an experimental study with a randomized and post-test-only control group design. Thirty male Wistar rats were divided into five groups: a normal control group, a negative control group given a high-fat, high-fructose diet, and treatment groups given Porang glucomannan 25mg, 50mg, or 100mg/200gBW. Twenty-eight days after the intervention, the SCFA level was measured using gas chromatography-mass spectrometry (GC/MS).

Results: The treatment group with Porang glucomannan 50 mg/200gBW has the highest mean SCFA level (3.98±0.83 nmol/ml) compared to normal control (1.56±0.24), negative control (2.18±0.45), treatment group 25mg/200gBW (1.81±0.26), and treatment group 100mg/200gBW (2.58±0.38). Kruskal Wallis test showed significant differences among groups (p<0.001). Post hoc test revealed that SCFA in the treatment group 50 mg was significantly higher than the normal control and negative control group.

Conclusion: Glucomannan from Porang (A. muelleri) at 50 mg/200gBW doses improves short-chain fatty acid in Wistar rats with high-fat and high-fructose diets.

Keywords: glucomannan, porang, short-chain fatty acid, wistar rat

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INTRODUCTION

The leading cause of death in humans is coronary heart disease [1]. The primary risk factor for coronary heart disease is abnormal blood lipid metabolism or dyslipidemia. Short-chain fatty acids (SCFAs) were shown in several studies to lower the body's levels of both total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglyceride. Zhao et al. reported that male hamsters fed with SCFA supplement for six weeks had reduced plasma total cholesterol by up to 17% [2]. Haghikia et al. found that treating high-cholesterol mice with SCFA significantly reduced their TC, VLDL, and LDL-C levels and stopped them from developing atherosclerosis [3]. A randomized controlled trial also revealed that giving SCFA orally for eight weeks to people with hyperlipidemia decreases several blood lipid components compared to the control group. These findings suggest that increased SCFAs would improve lipid profile and prevent coronary heart disease [4].

Considering the significance of SCFA for the body, it is imperative to search for alternative nutrition based on regional foods that might raise the body's SCFA levels. Glucomannan is one material that has been the subject of much investigation. Due to its potential health benefits, glucomannan is a popular study target, particularly for disorders associated with metabolic syndrome, such as dyslipidemia [5][6]. Glucomannan helps promote weight reduction by lowering blood pressure, glucose, cholesterol, and triglyceride levels. As a result, glucomannan-containing foods are considered healthier and widely available in Asian and European markets [7][8].

Glucomannan is a polysaccharide. That belongs to the group of soluble dietary fibers. High-viscosity glucomannan consumption decreases the absorption of bile acids in the ileum and cholesterol in the jejunum [9]. Additionally, glucomannan functions as a prebiotic, aiding in synthesizing GLP-1 and short-chain fatty acid biosynthesis. Lipid synthesis will eventually decline as a result of this process' impact on lipid metabolism [10]. Additionally, glucomannan influences the activation of the PI3K/AKT pathway, which is implicated in developing atherosclerotic plaque [11].

The porang plant, scientifically named Amorphophallus muelleri, is one of the native plants with the greatest concentration of glucomannan (64,98%-66,43%) [10][12]. A.muelleri is also known as tubers [8]. According to research by Danawati, administering A.muelleri flour with a glucomannan content of 61.82% at doses of 25, 50, and 100 mg/200gBW could stop mice fed with a high-fat, high-fructose diet from having higher cholesterol. However, there was no discernible difference between the three doses [12]. In a study by Safitri et al., the administration of A.muelleri flour, which has a glucomannan content of 66.43%, at doses of 25, 50, and 100 mg/200gBW in mice with metabolic syndrome resulted in a dose-dependent increase in HDL levels and a significant decrease in TC, LDL, and triglyceride levels [10].

Currently, glucomannan from A.konjac is sold as a dietary supplement, but A.muelleri has not been thoroughly investigated for food
and medicinal reasons despite the lower price and availability. Research on the potential of glucomannan in *A. muelleri* to increase SCFA and prevent dyslipidemia is still limited. This study investigated the effect of glucomannan from Porang (*A. muelleri*) on SCFA in Wistar rats with high-fat and high-fructose diets.

**METHODS**

**Study Design**

This experimental study used an animal model with a randomized post-test-only control group design. This research was conducted at the Integrated Biomedical Laboratory Unit, Faculty of Medicine, Udayana University Denpasar.

**Subject/Sample**

The experimental animals in this study were thirty male Wistar rats (*Rattus norvegicus*), aged 8-10 weeks and weighing 200-300 grams. A rat was eliminated if a veterinarian inspection revealed it was unhealthy (motionless) and did not want to eat.

**Interventions**

All samples were divided into five groups with six rats in each group: (1) K0: normal control group, given a standard diet for 28 days; (2) K1: negative control group, given a high-fat, high-fructose diet for 28 days; (3) P1: treatment group I, given a high-fat, high-fructose diet and Porang glucomannan 25 mg/200 g for 28 days; (4) P2: treatment group II, given a high-fat, high-fructose diet and Porang glucomannan 50 mg/200 g for 28 days; (5) P3: treatment group III, given a high-fat, high-fructose diet and Porang glucomannan 100 mg/200 g for 28 days. Randomization was performed by taking random numbers to indicate interventions. At the end of the intervention, all rats were still alive and were euthanized.

**Data Collection**

Porang (*Amorphophallus muelleri*) flour was obtained from PT. Portal Bali Sejahtera in Buleleng Regency, Bali, Indonesia. Examination of glucomannan levels showed that Porang flour has a glucomannan content of 24.93%. The high-fat diet was prepared by mixing the ingredients: standard feed 85%, pork fat 15%, duck egg yolk two cc. Pork fat was heated to the point of becoming oil. Crushed standard feed was combined with lard oil. The feed mixture was then dried in the oven. For 28 days, 20g of feed was provided daily. One duck egg yolk (up to two cc) was administered daily. A high-fructose diet was administered, 20% fructose syrup was mixed into drinking water, and ad libitum was given [13]. After 28 days of treatment, according to the assigned groups, mice were anesthetized using ketamine 100 mg/kgBW and xylazine 10 mg/kgBW intramuscularly. Blood samples were taken via the retroorbital plexus. Termination was carried out using the neck dislocation method. All rat carcasses were burned using an incinerator. SCFA in rat plasma was evaluated with gas chromatography-mass spectrometry (GC/MS) [14].
**Data Analysis**

All data analysis was performed in SPSS version 23. Data distribution was evaluated with the Shapiro-Wilk and Levene Test for homogeneity. The data was not normally distributed, so the Kruskal Wallis test was performed, followed by the post-hoc Dunn test. The assessment was conducted with a confidence interval (CI) of 95% and a p-value at the significance limit of 0.05.

**Ethical Consideration**

The experimental protocols were approved by the Faculty of Medicine Ethics Committee of Udayana University, with protocol number No.B/171/UN14.2.9/PT.01.04/2023 on 21st September 2023. The research was carried out according to the Helsinki Declaration rule.

**RESULTS**

There were 30 male Wistar rats, aged 8-10 weeks, weighing 200-300 grams, randomized into five groups. One rat in each group, P2, and P3, was excluded due to illness. Therefore, a total of 28 rats were included in the analysis. Rats in the treatment II group (given a high-fat high-fructose diet and Porang glucomannan 50 mg/200gBW) have the highest SCFA level among other groups (3.98 ± 0.83 nmol/ml), compared to normal control (1.56±0.24), negative control (2.18±0.45), treatment group 25 mg (1.81±0.26), and treatment group 100 mg (2.58±0.38) (Fig. 1).

Levene test showed that the data was homogenous (p=0.252), but the Shapiro-Wilk test showed that the data was not normally distributed (p=0.001), so the Kruskal-Wallis Test was used. Kruskal Wallis test showed significant differences in SCFA levels among groups (p<0.001). A post hoc Dunn test was then performed, showing a significant difference between normal control with treatment II and III but no difference with treatment I. The negative control was shown to have different SCFA levels with normal control and treatment II only. This result showed that glucomannan in Porang (*Amorphophallus muelleri*) at the dose of 50mg/200gBW improves short-chain fatty acid in Wistar rats with high fat and high fructose diets compared to normal control and negative control.
Fig. 1. Mean SCFA in each group. K0: normal control group; P1: treatment group I (Porang glucomannan 25mg/200gBW); P2: treatment group II (Porang glucomannan 50mg/200gBW); P3: treatment group III (Porang glucomannan 100mg/200gBW)

Table 1

SCFA level in Each Group

<table>
<thead>
<tr>
<th>Groups</th>
<th>Mean SCFA (nmol/ml)</th>
<th>SD</th>
<th>Min</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal control</td>
<td>1.56</td>
<td>0.24</td>
<td>1.22</td>
<td>1.91</td>
</tr>
<tr>
<td>Negative control</td>
<td>2.18</td>
<td>0.45</td>
<td>1.86</td>
<td>3.09</td>
</tr>
<tr>
<td>Treatment I</td>
<td>1.81</td>
<td>0.26</td>
<td>1.52</td>
<td>2.16</td>
</tr>
<tr>
<td>Treatment II</td>
<td>3.98</td>
<td>0.83</td>
<td>2.56</td>
<td>4.75</td>
</tr>
<tr>
<td>Treatment III</td>
<td>2.58</td>
<td>0.38</td>
<td>2.08</td>
<td>3.11</td>
</tr>
</tbody>
</table>
### Table 2

<table>
<thead>
<tr>
<th>Groups</th>
<th>P values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal control</td>
<td>0.042*</td>
</tr>
<tr>
<td>Treatment I</td>
<td>0.317</td>
</tr>
<tr>
<td>Treatment II</td>
<td>0.001*</td>
</tr>
<tr>
<td>Treatment III</td>
<td>0.003*</td>
</tr>
<tr>
<td>Negative control</td>
<td>0.042*</td>
</tr>
<tr>
<td>Treatment I</td>
<td>0.300</td>
</tr>
<tr>
<td>Treatment II</td>
<td>0.037*</td>
</tr>
<tr>
<td>Treatment III</td>
<td>0.290</td>
</tr>
<tr>
<td>Treatment I</td>
<td>0.317</td>
</tr>
<tr>
<td>Negative control</td>
<td>0.300</td>
</tr>
<tr>
<td>Treatment II</td>
<td>0.002*</td>
</tr>
<tr>
<td>Treatment III</td>
<td>0.041*</td>
</tr>
<tr>
<td>Treatment I</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Negative control</td>
<td>0.037*</td>
</tr>
<tr>
<td>Treatment II</td>
<td>0.002*</td>
</tr>
<tr>
<td>Treatment III</td>
<td>0.327</td>
</tr>
<tr>
<td>Treatment I</td>
<td>0.003*</td>
</tr>
<tr>
<td>Negative control</td>
<td>0.290</td>
</tr>
<tr>
<td>Treatment II</td>
<td>0.041*</td>
</tr>
<tr>
<td>Treatment III</td>
<td>0.327</td>
</tr>
</tbody>
</table>

### DISCUSSION

A high-fat, high-fructose diet is a risk factor for dyslipidemia and atherosclerosis. This alludes to earlier research that demonstrated that mice fed high-fat, high-fructose diets developed dyslipidemia. The egg yolk diet induces persistent hyperlipidemia, accumulating arteries lipids and promoting atherosclerosis. Fructose impairs glucose and lipid metabolism, resulting in increased visceral adipose tissue deposition, hepatic triglyceride accumulation, and insulin resistance; hence, a high-fat and fructose diet is recommended to create an animal model with dyslipidemia [10][13].

The glucomannan level of porang flour in this study, which used porang flour from Bali, was 24.03%. This level is lower than the glucomannan content using porang flour from Java, as shown by research by Danawati and Safitri et al., whose glucomannan content was above 60% [10][12]. In research by Mansniawati et al. using porang flour from several areas in South Sulawesi, the glucomannan level in porang flour varied around 18.01-24.12% [15]. Research by Qur'ani et al. in three regions of East Java showed that the glucomannan level in medium plains is 47.7%, greater than the glucomannan level in Porang from lower plains (22.8%) and higher plains (41.6%) [16]. Despite the lower glucomannan content in this study, this level of glucomannan still has a beneficial effect on SCFA. The glucomannan levels in Porang depend on the harvest time and the plantation area.
Glucomannan levels tend to be higher in Porang tubers harvested when the plant has fallen compared to glucomannan in tubers obtained before the plant has fallen. To get tubers with high levels of glucomannan, it is recommended to plant porang in the area at an altitude of 613 m above sea level [17].

Another glucomannan that was used as a commercial supplementation is Konjac glucomannan. Porang glucomannan exhibited better solubility (86.4%) and degree of acetylation (13.7%) than commercial glucomannan but poorer viscosity (5400 cps), WHC (34.5 g/g) and DP (9.4). Supplementation of Porang glucomannan in Wistar rats resulted in higher total SCFA levels and similar bacterial growth compared to Konjac glucomannan in a study by Harmayani et al. [18].

This study found that glucomannan from Porang (Amorphophallus muelleri) at 50mg/200gBW doses increased SCFA in Wistar rats with high-fat and high-fructose diets compared to normal and negative control. Glucomannan from Porang at a dose of 100mg/200gBW resulted in a lower SCFA level than a 50mg/200gBW dose, probably because the number of calories given increases with the glucomannan intake. High glucose due to excessive doses of glucomannan may consume more SCFA, which results in a lower level of serum SCFA, such as in this study. Through Ffar2 and Ffar3 pathways, SCFAs can raise PYY and GLP-1 expression in the colon. It has been demonstrated that PYY improves glucose absorption in muscle and adipose tissue, whereas GLP-1 causes the pancreas to produce less glucagon and more insulin. Furthermore, it has been demonstrated that SCFAs reduce hepatic gluconeogenesis by raising AMPK activation and phosphorylation. In addition, the transport of SCFA across the apical and basolateral membranes of the gut epithelial cells was performed mainly via active transport. Therefore, there is a limitation of SCFA that can be taken from the colon into the systemic circulation [19].

This study is in line with a study by Anggela et al. that stated that Porang glucomannan in fecal batch culture fermentation might increase SCFA, especially the butyric acid concentration than control (39.94 ± 7.67 vs 15.76 ± 0.40 mM) [20]. In research conducted by Perdinan and Larasati, Porang glucomannan supplementation in broiler chickens increased the total SCFA content in the jejunum. This is related to the prebiotic properties of glucomannan. Chicken lacks enzymes capable of inhibiting β-glucosidase in glucomannan, making it a potential prebiotic [21]. In a study by Itha’atur, the consumption of jelly containing Porang glucomannan for eight weeks increased beneficial bacteria Bacteroidetes, SCFA, and SIgA compared to placebo in the feces of adults with obesity [22]. In a study by Harmayani, a diet supplemented with Porang glucomannan in vivo inhibited the growth of Escherichia coli, increased the production of total SCFA, and decreased the pH of cecal content [18].

The mechanism of porang glucomannan to increase SCFA is attributed to the prebiotic effect of glucomannan. Glucomannan is a dietary fiber that may withstand digestion in the upper digestive tract, allowing it to pass through into the colon and be fermented by
healthy bacteria such as *Bifidobacterium* or *Lactobacillus* into SCFA including acetic, butyric, and propionic acid, which the colon absorbs. SCFA synthesis is essential in supplying energy to epithelial cells, boosting colon health, and improving intestinal immunity. Acetic acid is predominantly an SCFA that may be absorbed and carried straight into systemic circulation for lipogenesis, whereas propionate is transferred to the liver for gluconeogenesis. Butyric acid is claimed to have the butyrogenic effect that selectively stimulates the growth of bifidobacteria [20].

Short-chain fatty acids may inhibit the hydroxymethyl GLUTaryl-CoA reductase (HMG-CoA reductase) enzyme, which plays a role in cholesterol synthesis [23][24]. Short-chain fatty acids may also contribute to regulating body weight by promoting the release of hormones that promote satiety, such as GLP-1 and peptide YY (PYY). GLP-1 increases HDL by modifying the reverse cholesterol transport mechanism [25][10].

**IMPLICATION**

For health practitioners, understanding the implications of Porang glucomannan's effect on SCFA production can enhance dietary recommendations and therapeutic strategies for patients with dyslipidemia. Although more human research is required to corroborate these findings, Porang glucomannan, a local product, can be recommended as a dietary supplement to manage dyslipidemia and prevent coronary heart disease.

**STUDY LIMITATION**

The limitation of this study is the absence of a precise strategy for inducing a high-fat, high-fructose diet. The use of lard with a strong aroma may suppress the rat’s hunger, while only a restricted amount of egg yolks could be given due to the limited capacity of the rat's stomach. It is necessary to modify the high-fat, high-fructose diet by adding food flavorings to the feed so that the aroma of pork fat does not overly influence the rats' appetite. Aside from that, additional methods of delivering fructose might be investigated to ensure that the amount of fructose taken is accurately and uniformly distributed across each group. This study did not investigate the type of SCFA or explore mouse activity or other variables, such as hormones and genetics, that may impact rat SCFA. Last, a supplementary diet such as Porang glucomannan needs high adherence to produce a positive effect, which is hard to achieve outside of laboratory conditions [26].

**CONCLUSION**

Glucomannan from Porang (*Amorphophallus muelleri*) at 50mg/200gBW doses improves short-chain fatty acid in Wistar rats with high-fat and high-fructose diets. Additional research was required to determine the impact of Porang on human health indices, including blood pressure, blood glucose levels, lipid profiles, weight, and insulin resistance.

**CONFLICT OF INTEREST**

The authors declare that there are no conflict of interests related to the study.
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